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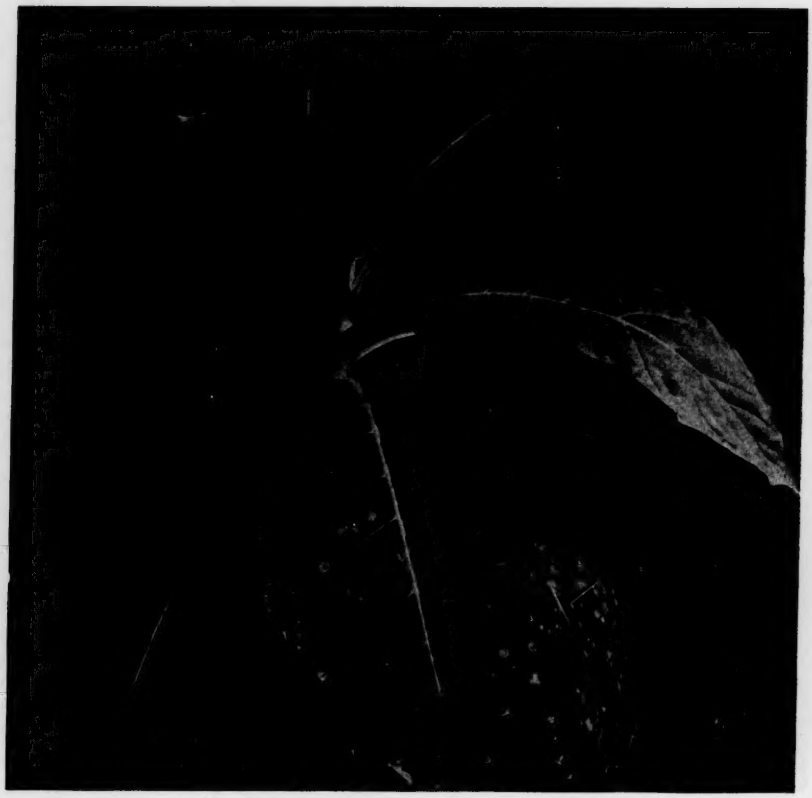

MOSAIC RESISTANT TOBACCO

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New Data on Rh and Feeblemindedness

Hereditary Ataxia

The American Quarter Horse

VOLUME 36 NOVEMBER, 1945 NUMBER 11

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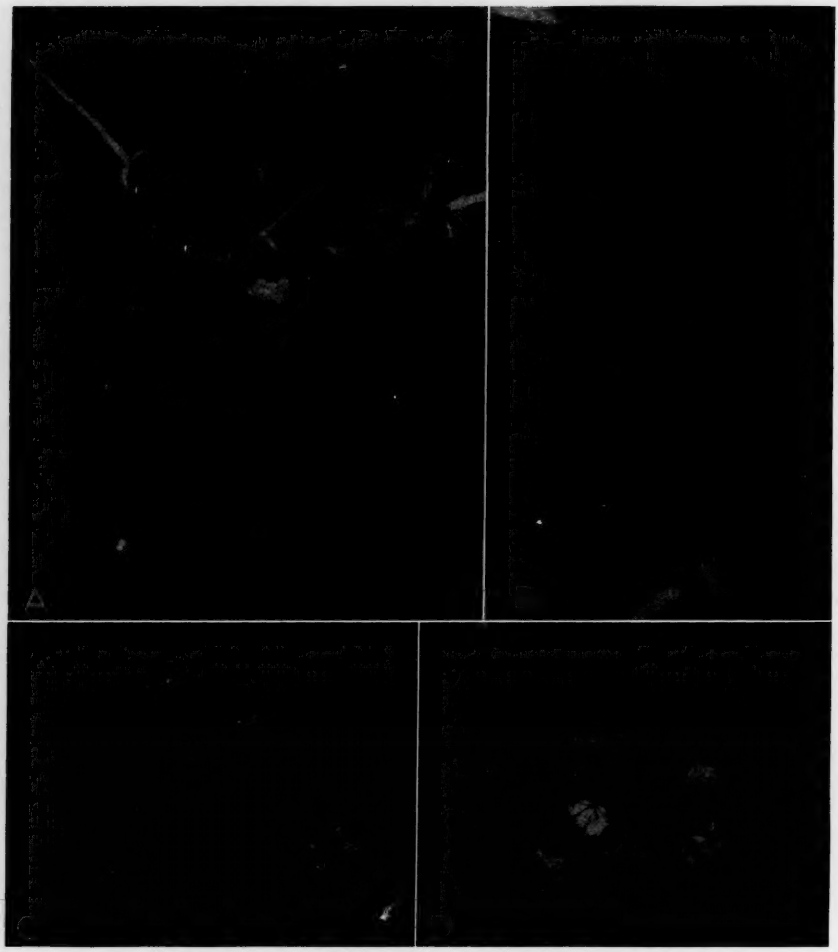

MOSAIC AND NECROSIS IN NICOTIANA GLUTINOSA AS AFFECTED BY TEMPERATURE

Figure 1

All plants were inoculated with the virus of tobacco mosaic. *A* and *B* show mosaic mottling at 97° F. Leaf marked with pin was wiped with virus; note the chlorotic local spots and rings. *C* shows necrotic local lesions only at 75° F., and *D* shows the lethal effects of secondary necrosis at 80° F.

GENOTYPE AND TEMPERATURE IN RELATION TO SYMPTOMS CAUSED IN NICOTIANA BY THE MOSAIC VIRUS

Studies on the Necrosis and the Mottling Reactions in Certain Species of *Nicotiana* and in Tobacco Hybrids Infected With the Tobacco-Mosaic Virus

H. H. McKINNEY and E. E. CLAYTON*

EXTREME variation in the expression of local and secondary necrosis in species of *Nicotiana* and in breeding lines of tobacco carrying the necrosis factor (*N*) has made it necessary to obtain some estimation of environmental influences. Many observations indicated that temperature is very important. As in species of *Capsicum* carrying the *N* factor,[8] it became evident that the presence of certain genes regulating mosaic expression could not be detected, except under extreme conditions of temperature.

Methods

All plants were inoculated by wiping the leaves with extracts of the wild or field type of tobacco common-mosaic virus, *Marmor tabaci* (Holmes ex Valleau) McKinney.[7] As virus necrosis is likely to be lethal in very young plants of some species at 70° F., the plants were always allowed to grow beyond this stage before they were inoculated. However, *Nicotiana benthamiana* Domin and *N. repanda* Lehm were very susceptible to the lethal reaction even in the later stages of growth. Plants were cultured in pots of ample size in greenhouses and in culture chambers. When a species or hybrid carrying the *N* factor developed mosaic or other unusual symptoms, isolations and tests were carried out to make certain that the etiological agent was common-mosaic virus and not some other species or strain.

Results

The reactions of several species of *Nicotiana* when cultured at different temperatures, are listed in Table I. These results show that: (a) in some of the species part of the populations carried the mosaic factors only; (b) necrosis was not expressed in the same manner in all species at a given temperature; (c) in *N. glutinosa,* the temperature range for secondary necrosis was more restricted than it was in the other species tested; (d) each species, with the possible exception of *N. repanda,* carries one or more factors for mosaic susceptibility the presence of which is apparent only at temperatures which are above those favoring severe secondary necrosis. Symptoms in *N. glutinosa* and *N. rustica* are illustrated in Figures 1 and 2.

Similar tests with *Nicotiana tabacum* × *N. glutinosa* hybrids have exhibited differences in the expression of necrosis at a given temperature, and all such hybrids that have been tested, have manifested mosaic mottling at temperatures above the optimum for severe secondary necrosis (Figure 3).

All species and hybrids carrying the *N* factor, and which had mosaic at high temperatures, became necrotic when the temperature was lowered to 75° F. or below, and sometimes the plants were killed. The variety of *Nicotiana rustica* studied usually survived this drop in temperature, and many secondary lesions developed on the foliage and stem, just as when the plants were cultured at 75° F. throughout a test.

In a note[2] it was pointed out that tobacco carrying the *N* factor from *Nicotiana glutinosa* is sometimes killed in the

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MOSAIC AND NECROSIS IN NICOTIANA RUSTICA AS AFFECTED BY TEMPERATURE

Figure 2

A shows plant infected with virus of tobacco mosaic, and cultured near 95° F. *B* shows similarly infected plant cultured near 75° F.; note necrotic local lesions and lethal effects of secondary necrosis. *C* shows mosaic leaf from plant *A*.

field during hot weather when infection takes place in plants that are well advanced beyond the seedling stage. For this reason the writers questioned the practical value of crosses between *N. glutinosa* and the commercial types of mosaic-susceptible tobacco (*N. tabacum*). However, when it became apparent that *N. glutinosa* also carried a mosaic factor, and that the *N* factor and the mosaic factors in other species so greatly differed in their expressions with changes in temperature, our interest turned to the use of parents in which the factors for mosaic mottling and virus production are reduced to a minimum. Ambalema tobacco carries two recessive factors (a_1a_1 a_2a_2) for mosaic resistance.[1] T. I. 448 tobacco is more resistant than Ambalema,[5] in that it is a less favorable host for virus production, and it manifests no visible symptoms under ordinary culture conditions. Its genetic constitution, with respect to mosaic resistance, may be regarded as a_1a_1 a_2a_2 ++. At 92° F. both of these tobaccos developed a few faint "oak-leaf" patterns,[6] which indicate the presence of factors of a low order for mosaic. However, the typical mosaic mottling that is associated with leaves when invaded at a very early stage, has not been induced in these genotypes when tested over a wide range of temperatures.

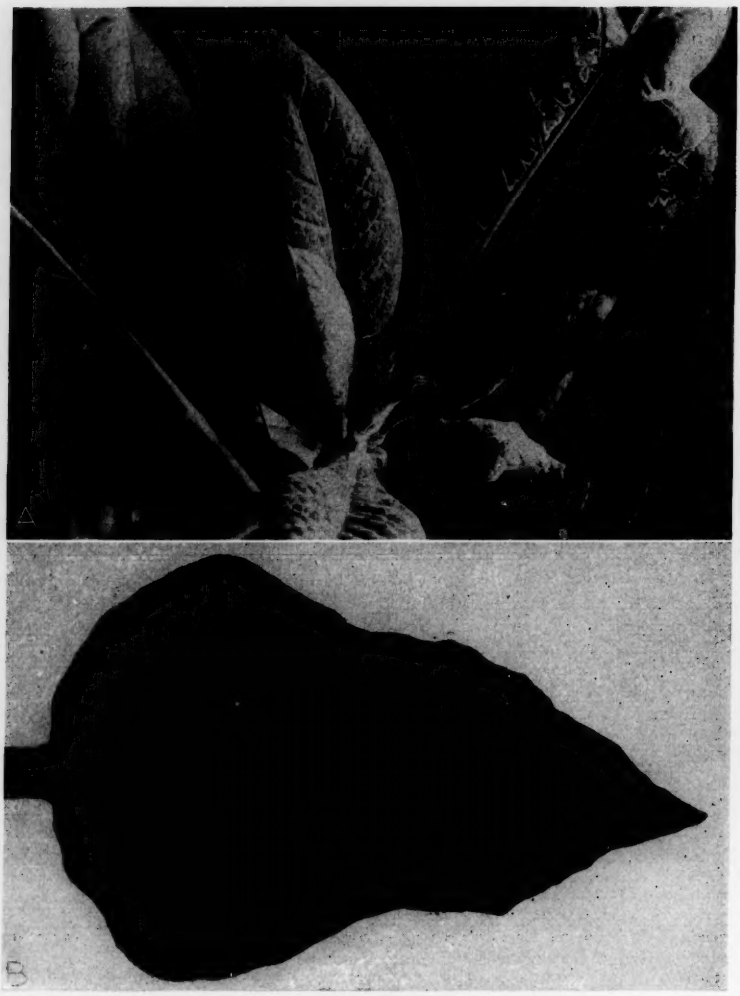
Hybrids Involving Burley, Ambalema and Glutinosa

A Burley tobacco genotype, homozygous for the necrosis factor *N* was crossed with homozygous Ambalema-Burley (*nn* a_1a_1 a_2a_2) and the F_1 backcrossed to Ambalema-Burley. A 1943 field selection (No. 20) gave a local necrotic response in the greenhouse (fall of 1943) and seed (F_2) of this was planted in the field in 1944, and necrotic reacting plants were selected. Three flowers of each selection were back-

TABLE I.—Reactions of seven species of *Nicotiana* when inoculated with tobacco-mosaic virus and cultured at different temperatures.

	Culture temperatures			
	60° F.	72.5° F.	97° F.	102° F.
N. glutinosa L. (1)	Local necrotic lesions	Local necrotic lesions	Vein clearing, secondary diffuse chlorosis and mosaic	Vein clearing, mosaic
N. rustica var. *brasilia* Schrank	Local necrotic lesions	Local necrotic lesions, secondary necrosis	Local chlorotic lesions, vein clearing, mosaic	Local chlorotic lesions, vein clearing, mosaic
N. alata Link & Otto (2)	Local necrotic lesions	Local necrotic lesions, secondary necrosis	Local chlorotic lesions, vein clearing, mosaic	. . . . .
N. sanderae W. Wats (2)	Local necrotic lesions	Local necrotic lesions, secondary necrosis	Local chlorotic lesions, vein clearing, mosaic	. . . . .
N. maritima H.-M. Wheeler (2)	Local necrotic lesions	Local chlorotic lesions, secondary necrosis	Local chlorotic lesions, vein clearing, mosaic	. . . . .
N. benthemiana Domin	Local scalds and necrotic lesions. Mild secondary necrosis	Local scalds, secondary necrosis	Local chlorotic lesions, secondary necrosis	Local necrosis and chlorotic lesions. Vein clearing, mosaic
N. repanda Lehm.	Local necrotic lesions. Mild secondary necrosis	Local necrotic lesions. Mild secondary necrosis	Local chlorotic lesions, secondary necrosis	Local chlorotic lesions (3)

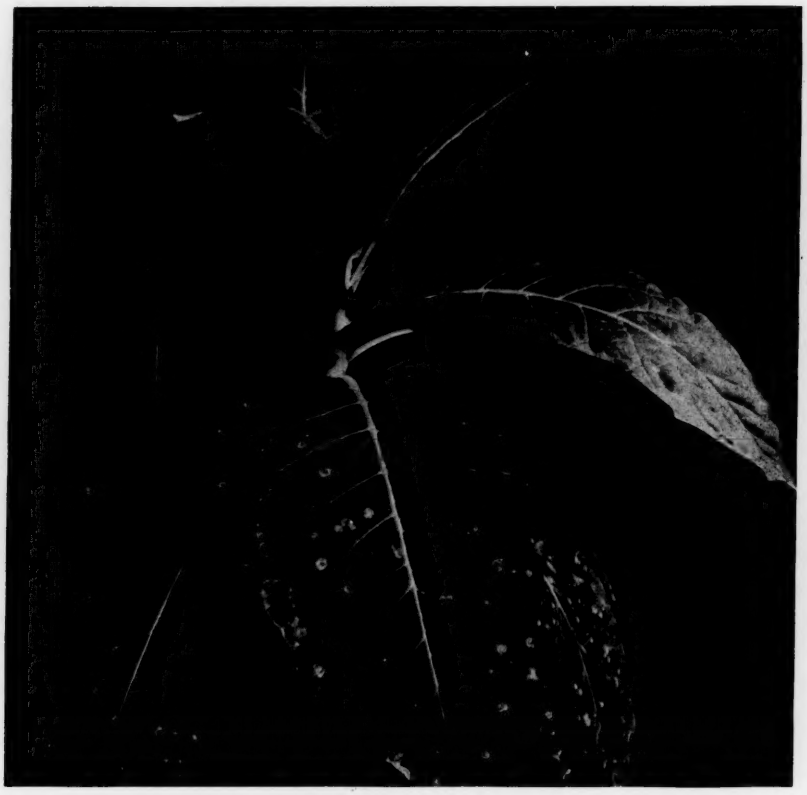
(1) Additional tests with *N. glutinosa* indicate that mosaic usually appears when the temperature is held near 92° F., but secondary necrosis occurs between 85° and 90° F.
(2) A portion of the population carried the mosaic factors only.
(3) Two plants developed secondary necrosis, 3 plants were free of secondary symptoms. Similar results were obtained in another test conducted at 106° F.



MOSAIC IN TOBACCO CARRYING THE N FACTOR

Figure 3

A—Tobacco plant with genetic constitution *Nn* A_1A_1 A_2A_2. This plant was inoculated with tobacco-mosaic virus, and cultured near 88° F., at which temperature it developed mosaic. The temperature was lowered to 75° F., and within 24 hours the necrotic "oak-leaf" pattern appeared on the large leaf, and within five days the plant was dead from secondary necrosis. *B*—Mosaic in a leaf of Samsun-type tobacco with genetic constitution *NN* A_1A_1 A_2A_2. This plant was inoculated with tobacco-mosaic virus, and cultured near 88° F.



SECONDARY LESIONS IN TOBACCO

Figure 4

Burley-type tobacco with genetic constitution $NN\ a_1a_1\ a_2a_2$, inoculated with tobacco-mosaic virus, and cultured at temperatures ranging from 75° F. at night to 95° F. and above during the day. Chlorotic and necrotic lesions appeared on the inoculated leaves, but the only secondary symptoms to appear were the chlorotic-necrotic spots and rings illustrated.

crossed again to Ambalema-Burley, and the remaining flowers were selfed to give F_3 seed. Seven-week-old seedlings of these lots were inoculated by wiping the leaves with an extract of virus, and tested in the greenhouse at 70° F. during the fall of 1944. All 20 test plants of the F_3 line (20-14) gave the local necrotic response, and all of the 20 plants of the line backcrossed to Ambalema-Burley also showed local necrotic reactions. None of the plants showed any symptoms for mosaic. Therefore, it appears certain that the F_3 line No. 20 was genetically $NN\ a_1a_1\ a_2a_2$ and that the F_1 backcross to Ambalema-Burley was $Nn\ a_1a_1\ a_2\ a_2$.

Plants of the F_3 line were inoculated again and tested at 75° and 90° F. with the following results: At 75° there were many local necrotic lesions. There was no secondary necrosis and no mosaic. Assays on Scotia beans revealed traces of systemic virus in new leaves of some, but not all of the plants. As the spring season approached, the greenhouse temperatures increased to an

MILD MOSAIC IN TOBACCO CARRYING THE N FACTOR IN COMBINATION WITH FACTORS FOR MOSAIC RESISTANCE

Figure 5

Leaves from plants infected with tobacco-mosaic virus, and cultured near 90° F. for 34 days, followed by 75°; showing mild mosaic and necrotic patches. *A* shows leaf from a plant with genetic constitution $NN\ a_1a_1\ a_2a_2$. Mild mosaic appeared at 90°, the necrosis appeared within 24 hours after the temperature was lowered to 75° F. *B* shows leaf from a plant with genetic constitution $Nn\ a_1a_1\ a_2a_2$ ++. Mild mosaic and necrosis appeared within three to six days after the temperature was lowered to 75°.

average near 80°, with day temperatures sometimes exceeding 95° F., yet no secondary symptoms appeared in these tobacco plants. When leaves on these plants were inoculated by wiping with an extract of tobacco-mosaic virus during this warm period, many chlorotic and necrotic local lesions appeared, the virus entered the new leaves, and induced chlorotic spots and rings that later became necrotic as illustrated in Figure 4. No necrosis appeared in the stem or growing tip after this inoculation. Tobacco-mosaic virus was the only one recovered from the secondary ring spots.

In later tests with this F_3 line, it was observed that infected plants develop secondary, faintly chlorotic spots at temperatures near 77° F., and as the temperature was raised to 80° and 85° F., the spots became increasingly chlorotic. There was no necrosis in the stem. Like the faintly chlorotic spots appearing in the leaves of Ambalema tobacco plants, these spots did not appear until the leaves were fairly well developed.

At 90° F., weakly chlorotic and necrotic local lesions, and a weak but distinct mosaic pattern developed in a few leaves on each plant, but there was no secondary necrosis. Assays on Scotia beans revealed a high concentration of

THREE INFECTED TOBACCO HYBRIDS

Figure 6

Plants of the same age infected with virus of tobacco mosaic, and cultured near 80° F. *A* shows plant with genetic constitution $Nn\ a_1a_1\ a_2a_2\ ++$. *B* shows Burley-type plant with genetic constitution $NN\ a_1a_1\ a_2a_2$. *C* shows Burley-type plant with genetic constitution $NN\text{-}A_1A_1\ A_2A_2$; note secondary necrosis and lethal effects. Plants *A* and *B* had secondary, faintly chlorotic spots, but no necrosis or mosaic. All plants developed chlorotic lesions in the inoculated leaves.

systemic virus in the leaves. After 34 days at 90° the temperature was lowered to 75° F. Within 24 hours necrosis appeared in the upper part of the stem and in the mottled leaves. As the plants developed, the mottled leaves (Figure 5*A*) and one to three leaves below these became severely chlorotic and necrotic, and a few small chlorotic spots appeared in two to three of the new leaves. Subsequent leaves were free of symptoms, and the stem necrosis was very mild, being confined chiefly to the outer cortex, and not involving the growing tip. When several symptom-free leaves had developed they were inoculated by wiping with an extract of tobacco-mosaic virus. Many chlorotic and necrotic local lesions appeared. Virus entered the leaves subsequently developed and induced yellow spots and rings (Figure 4) that later became necrotic. When this inoculation was made the greenhouse temperatures averaged near 80° F., with the day temperatures sometimes exceeding 95° F.

Hybrids Involving Burley, Ambalema, T.I. 448 and Glutinosa

In conjunction with the Ambalema-Burley material, studies have been made with combinations of the *NN* genes from *Nicotiana glutinosa* with T.I. 448 tobacco. This latter genotype has both pairs of Ambalema genes with additional modifying gene or genes that make it more highly resistant to the multiplication of the virus. Up to the present, we have conducted infection tests with F_2 plants from a Burley-glutinosa (NN) $\times$ Ambalema ($nn\ a_1a_1\ a_2a_2$); necrotic reactor (F_1) backcrossed to Ambalema; necrotic reactor backcrossed to T.I. 448; necrotic reactor selfed to provide a test of the F_2, and backcrossed again to T.I. 448 to provide a test of the F_1. Necrotic plants from the F_2 and the F_1 generations were tested at different temperatures.

The F_2 plants had the genetic constitution $a_1a_1\ a_2a_2$, but pending further testing it is uncertain whether they are Nn or NN, and also whether they have the

modifying factors for virus infection from the T.I. 448 parent.

The F_1 plants are definitely $Nn\ a_1a_1\ a_2a_2\ ++$. Twenty plants were inoculated and cultured at 70° F. Of these, six developed necrotic lesions and 14 remained healthy. Plants free of local lesions were continued at 75° and remained free of mosaic and chlorotic spotting. The plants which developed local necrotic lesions at 70° were reinoculated by wiping virus on new leaves, and tested at 75° and 90° with the following results: At 75° local necrotic lesions developed, but there was no secondary necrosis, and there was no mosaic. Assays on Scotia beans revealed slight traces of systemic virus in the leaves of some, but not all of the plants. At 90° the plants were free of local and secondary necrosis and mosaic. However, at this high temperature, a few very inconspicuous secondary light-green spots appeared in a few of the leaves on a few plants. Assays on Scotia bean plants revealed much systemic virus in the leaves of all plants.

After 34 days at 90° F. the temperature was reduced to 75°. Within three days a few necrotic spots appeared on a few of the upper leaves known to contain virus, and three days following this observation, mild light-green mosaic appeared in three to four upper leaves on each plant (Figure 5*B*). The mosaic patterns resembled the "oak-leaf" and patch patterns associated with tobacco ring-spot virus, some of the legume-mosaic viruses and tobacco-mosaic virus in leaves that are invaded late in their development. Leaf necrosis developed in much the same manner as in the Burley-type plants described above, except that new leaves were completely free of spots and all other signs. There was no sign of necrosis in the stems.

In another test, plants of the same genetic constitution were inoculated and cultured near 80° F. The primary and secondary symptoms consisted of weakly chlorotic spots. There was no necrosis in the stem and no mosaic. Infected plants of three genetic constitutions, and cultured near 80° F. are illustrated in Figure 6.

Comparison Between Certain Hybrids in Relation to the Local Necrotic Lesion Reaction

Observations on field tests indicated that inoculated Burley-type hybrids carrying the *N* factor and the Ambalema factors developed smaller local necrotic lesions with a longer incubation period than was the case with hybrids carrying the *N*, but not the Ambalema factors.

In a test conducted in the greenhouse at temperatures near 75° F., two Burley-type hybrids carrying the *N*, but not the Ambalema factors, developed local necrotic lesions measuring from 2-5 mm. in diameter. The incubation period was 40-50 hours. In the same test, two Burley-type hybrids carrying the *N* and the Ambalema factors, also two T.I. 448-type hybrids carrying the *N* factor, developed local lesions measuring from one to two mm. in diameter. The incubation period was 65-75 hours.

The *NN* and the *Nn* constitutions appear to be very similar, if not alike, with respect to their incubation periods and their lesion-induction characteristics. Holmes[4] noted no differences between the responses of his *NN* and *Nn* plants.

Discussion and Conclusions

The so-called glutinosa-type of resistance appears to be determined by a gene-controlled temperature-response mechanism which regulates the expression of the necrosis and the mosaic factors present.

The behavior of *Nicotiana repanda* suggests that the necrosis factor may be isolated from the mosaic factors. However, in this species, secondary necrosis occurs at moderate culture temperatures. Attempts to effect a cross with *N. tabacum* have not been successful.

Any economic value that may accrue from the new hybrids described cannot be predicted. However, it is apparent that, (a) factors for secondary necrosis in the stem do not operate or are absent in the T.I. 448 hybrids carrying the *N* factor, and they come to expression in an unusually weak form in the Burley-type hybrids carrying the Ambalema fac-

tors; (b) factors for mosaic expression are present in both hybrids, but other complicating genetic factors that regulate the response to temperature, prevent their expression under the usual conditions of culture; (c) in both hybrids under discussion, the mosaic expression is unusually weak at the favorable high temperatures, indicating that some factors present in earlier hybrids have been eliminated or that suppressing factors have been introduced.

Irregularities in chromosome behavior introduced with the *N* factor from *Nicotiana glutinosa* must be kept in mind as Gerstel's[3] study of hybrids from *N. tabacum* × *N. glutinosa* indicates that not merely the *NN* gene, but the entire chromosome carrying these genes has been transferred from *N. glutinosa* to *N. tabacum*. Consequently homozygous necrotic reacting genotypes should have 23 pairs of *N. tabacum* and one pair of *N. glutinosa* chromosomes. The full effects of the *N. glutinosa* chromosome remain to be determined. On the other hand, hybrids derived from crosses between the mosaic resistant (Ambalema or T.I. 448) and the mosaic susceptible commercial varieties of *N. tabacum* involve only *N. tabacum* chromosomes, and here the problem of possible injury to quality and yield from factors derived from another species does not arise.

Summary

1. Seven species of *Nicotiana* carrying the necrosis factor (*N*) were inoculated with the tobacco-mosaic virus, and cultured at several temperatures.

2. With the possible exception of *Nicotiana repanda*, each species carries one or more factors for mosaic susceptibility the presence of which is apparent only at temperatures that are above those favoring severe secondary necrosis.

3. When the *N* factor was tranferred to plants carrying the mosaic-resistance factors from Ambalema and T.I. 448 tobaccos, secondary necrosis at high temperatures was greatly reduced or eliminated, and mosaic was greatly reduced.

4. Since it appears that the transfer of the *NN* gene from *Nicotiana glutinosa* to *N. tabacum* involves an entire chromosome, it is yet not clear that lines carrying the *N* factor are superior to the lines carrying the mosaic-resistance factors from Ambalema or T.I. 448 tobaccos.

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BREAD WITHOUT STONES

THE miller and the baker both play essential parts in producing the staff of physical life, and it is idle to argue which is the more important: if we want bread we need both. The miller of phenomena, who grinds his data to powder, and the baker of the bread of knowledge, who interprets the relationships of facts and hypotheses, both are needed if we are ever to evolve an effective and integrated social organism. It may be that we have been passing through a cycle of too finely grinding our facts. The specialists have been having a field day of compartmentation and differentiation. This has been viewed with alarm by those who see real danger in too cyst-like specialization to a healthy growth of biological research. The broader implications of science in affecting the cultural relationships of human beings have been even more neglected. Not only the man in the next cubicle of specialization, but the man in the street

urgently needs to know how research is faring, and also what it means in terms of values. In the age of the Atomic Bomb this assumes a new urgency.

A gallant attempt to bring integration out of this chaos of encystment has recently been made by a "miller" who bakes the powdery flour of fact into loaves which can nourish understanding. This miller has a name to conjure with in biological circles—H. J. Muller. The "H." is for "Herbert," not "Herman." He is a professor of English, not a geneticist. The reviewer reports that the relationship between these two H. J. Mullers is not that of identical twins. They are cousins.

His book* attempts to bring together the different viewpoints of enlightened artists, religionists and scientists, and to synthesize from the scientists' life-facts and Universe facts a solvent capable of dissolving the all too impenetrable partitions into which our modern world has become divided. Professor Muller believes that the compartmentation is far more serious than are the incompatibilities of tradition and concept characterizing the occupants of the various compartments:

More serious than the notorious "conflicts", between science and literature or religion and business, is the *separation* of these major interests. Cross-purposes do cross, and so may conceivably be reconciled; isolate purposes may never meet at all. A specialization in the interests of freedom and efficiency has in fact led to bureaucratic narrowness and waste. Hence John Crowe Hansom describes this godles, loose-laced age as "Puritan." Religion becomes Protestant, divorced from ritual and art as from the state; business preaches the gospel of *laissez-faire;* art also aspires to autonomy, in the name of "purity"; science becomes positivist and dismisses as "meaningless" the vital questions it cannot answer. All resist interference by other interests, resist the demand for integration. Keep the government out of business, keep religion out of public affairs, keep public affairs out of science, or art, or education—"Keep every thing out of everything else," writes Kenneth Burke, "and then complain that things are in pieces." "Gross materialism" is the common label for our condition, but "pure idealism" would stick as well. Religion, philosophy, science, or art for its own sake is finally as narrow and unillumined as business for its own sake. They are alike monopolists of capital, to use John Dewey's words: "and the monopolization of spiritual capital may in the end be more harmful than that of material capital."

Professor Muller is a master of the well-turned phrase, and writes with a saltiness that is delightfully challenging and refreshing. He has read widely and pondered deeply over ways and means to reassemble the rather badly battered temple of the 1945 Universe. He wrote before The Bomb further shattered the foundations of the Best of All Possible Worlds. The need to do this remains the same, even were this actually an age of science, for "despite their scientific or sophisticated manner, men have the same hankering as their pious ancestors for a cozy Universe, a closed system of certainties erected upon a single principle." They still pretend this is possible, even as the fragile green glass from the New Mexico crater appears in New York jewelry stores.

Commenting on the scientific scene he has this to say:

My use of the word "reason" is qualified accordingly. A better word for my purposes would be "intelligence"—except that it has already been brought into disrepute by psychologists with a rage to measure something before they have defined what it is they are measuring. ("Intelligence," Thorndike once remarked, "is the thing that psychologists test when they test intelligence.") What is needed, under any name, is a view of the biological whole man, a view in which we can make out the full value of the rational but also the necessity of the non-rational—feeling, emotion, sentiment, desire.

Unquestionably, science is dangerous. So are all adventures in thought, all dealings with the world; so is the whole experiment of civilization.

The greatest victories of science have in a sense been cheap victories, for they have been won by a careful refusal to engage the more difficult, certainly no less important problems of ethics, politics, and the humanities generally. The physicist rules out all that he cannot rule, reduces what is left to the simplest possible terms. It is an excellent policy—for his purposes. But he may then forget that what cannot be answered or calculated may never-

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*Muller, Herbert J. *Science and Criticism, the Humanistic Tradition in Contemporary Thought.* Pp. XIV, 305. $3.75. Yale University Press. 1944.

theless be immeasurably important, have incalculable consequences, and that the terms of life as it is lived are not simple.

In attempting to draw out the implications of the new concepts in physics, one has first to forego a deal of pleasing phantasy. Literary men have been inspired particularly by the Fourth Dimension: a nominal definition or mere convention of measurement that has become a symbol of all the magic of science. Thus we have J. B. Priestley (after J. W. Dunne) romping along this dimension into a new kind of Immortal Reality, reveling in a new kind of bed-time story, in the delusion that Einstein has paid for his ticket. One might remark that Priestley is a piker; if he likes extra dimensions he should turn to quantum physics, where the possible positions of just ten bits of matter form a thirty-dimensional continuum—and so he could go on indefinitely in this three-for-one ratio. But these ingenuous fables are harmless only when they are taken for fable.

The biologist as prophet comes for a rather rough going over:

Similarly, however, much arrogant optimism has also been inspired by biology. As a supposed guarantor of Progress, the theory of evolution has become the investment trust for all easy faith . And biologists themselves often have wild dreams about what they may do for the race.

In the patriotic enthusiasm over biology's Declaration of Independence, there has naturally been considerable oratory. At times one gets the impression that biologists have cut all ties with the motherland of physics. . . . Biology and physics are more intimately related now than they actually were in the calmiest days of mechanism.

And debaters in distress still flee to the old havens. "You can't change human nature," they announce; and then there is no getting at them. They have formed an axis with Jehovah.

Man has clearly been able, in a brief hour of biological time, to make over both himself and his environment, to create new necessities. He has made such marks on nature, God help him, as a dust bowl. And the dust bowl, an altogether natural, lawful result of human behavior, is not an inevitable outcome of human existence; by as natural, lawful means man can deliberately repair the damage.

In spite of its big words, Professor Muller sees hope for the psychologist, too:

But [the hope of a science of psychology] is an intoxicating hope, and the extravagance of many psychologists is as understandable as their popularity. To the common man, all opportunity comes at the "psychological moment," no truth is so intimate and impressive as a "profound psychological truth"; it is hard for professionals in such truth not to exploit such moments. Specifically, however, the chief trouble remains the confusion over first principles. . . . Their main concepts—perception, trait, instinct, learning, insight, personality, etc.—are usually ambiguous; if they do give these terms a precise meaning, other meanings creep back from the public world. (Hence someone has defined psychology as the subject in which you talk about things which everybody knows about in terms which nobody understands.)

The behaviorists have failed to learn that the serious business of psychology, as of every science, begins at the level of concepts, not of raw facts, and that the concepts must be derived from the subject matter, not imposed upon it. For they are among the last survivors of the good old days, when all the world was real stuff, stuff without nonsense, and good sense lay in recognizing the unreality of all sense. They believe that they are ultrascientific because, like classical physicists, they have abstracted from their subject matter all its distinctively human qualities—the awareness of behavior that constitutes "experience."

Sigmund Freud could write, in all modesty, that he belonged with those who have "disturbed the world's sleep." His work has made a permanent difference in man's thinking about himself; the world will never again sleep quite so soundly, or consciousness regain its innocence and purity. . . . If one takes the whole story literally, troublesome questions arise about just who or what is being satisfied or fooled by all the fantastic subterfuges, and how the not too bright Censor got and holds his job, and what kind of satisfaction an individual gets from symbolically satisfying a desire he doesn't know he has.

If some special scientific knowledge was required for the control of human beings, Pratt observes, Hitler would still be an obscure house painter.

In short, the instincts of common discourse are less the cause than the result of social institutions: a man is acquisitive *because* he can own property and acquire prestige with it. As Dewey holds, man is not primarily a creature of instinct or of reason; he is a creature of habit. Contrary to the belief of conservatives, instincts are easier to educate and modify than are institutionalized habits. Contrary to the hope of radicals, such habits are extremely difficult to eradicate. Children never resist education or innovation as do civilized adults; they may be full of disgusting impulses but they have few settled habits. The still very general tendency to assume

inviolable laws of human nature, and to find these laws written in local customs, is itself evidence that the Idol of Idols—of the cave and of the tribe, in the theater and in the marketplace—is Habit."

Muller considers the artist to be the catylist who makes the social mechanism tick through giving man an insight as to his place in nature and a universe significance. But science has necessarily changed the perspective. The for-granteds on which our universe is based are, willy nilly, modified by what we know about the universe. Hence Part 2 is devoted to "Development, the implications of modern science." Here are considered the problems of what science is, the philosophy of modern phvsics, the world of Einstein, the world of the semanticist. Coming nearer home, we have chapters on biology, psychology, and the social sciences, and the place of the individual in society.

Part 3, entitled "Re-statement" attempts to fuse these points of view into a scientific humanism, and to relate this to philosophy, nature, religion, and the political order. This does not come to any set formulas: as how could it? "A horse," D. H. Lawrence sadly observes, "is always true to its pattern; but a man you never can rely on." It is precisely this lack of patterning, precisely the fact that human beings can exercise choice processes as no other organism is able to, that makes the game of creating an integrated Humpty-Dumpty out of the specialized fragments, and constructing a cozy universe and a workable democracy, a very fascinating venture. The critic, as Muller sees it, has a job to do in keeping the tangibles and the intangibles in their proper places, calling fables what they really are. And he believes this is a business of action, not of watchful waiting and sessile faith. "I believe that only by trying to think can one really get ready for thought. I prefer faith *with* works."—R. C.

A FURTHER NOTE IN THE Rh FACTOR AND FEEBLEMINDEDNESS

IN the January 1945 issue of this JOURNAL we reported the results of a study of the Rh factor in 66 mothers and their 68 feebleminded offspring. The proportions of Rh— mothers and of Rh+ children from Rh— mothers were excessively high, consistent with the findings of Yannett and Liebermann. During the autumn visiting days at the Ohio Institution for the Feebleminded we have been able to obtain blood from 47 additional cases of undifferentiated mental deficiency and their mothers. The high frequencies of Rh— mothers, and of Rh+ children from Rh— mothers, were not maintained in these additional instances. Of the 47 mothers, seven were Rh—, only slightly higher than expectation. Of the 47 children, five were Rh+ from Rh— mothers, again only slightly higher than expectation.

In our total results of 113 mothers and their 115 feebleminded children, 24 of the mothers are Rh—, and 16 of the children are Rh+ from Rh— mothers. These results are still in excess of those expected, but the chi square values are much lower than those previously reported, and the deviation in regard to Rh+ children of Rh— mothers is now only significant instead of highly significant.

Needless to say, we shall continue to gather material as rapidly as possible.

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HEREDITARY ATAXIA

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ATAXIA, although a rare condition, has been reported in a number of families. We are presenting a new kinship in which 18 males and two females have an ataxic type of walk. Eighty-two males and 80 females are recorded in the six generations of this family. There are four medical doctors in this kinship who have observed the condition through four generations. Information concerning the various members in the families has been obtained by the junior author through conversation or correspondence with the individuals.

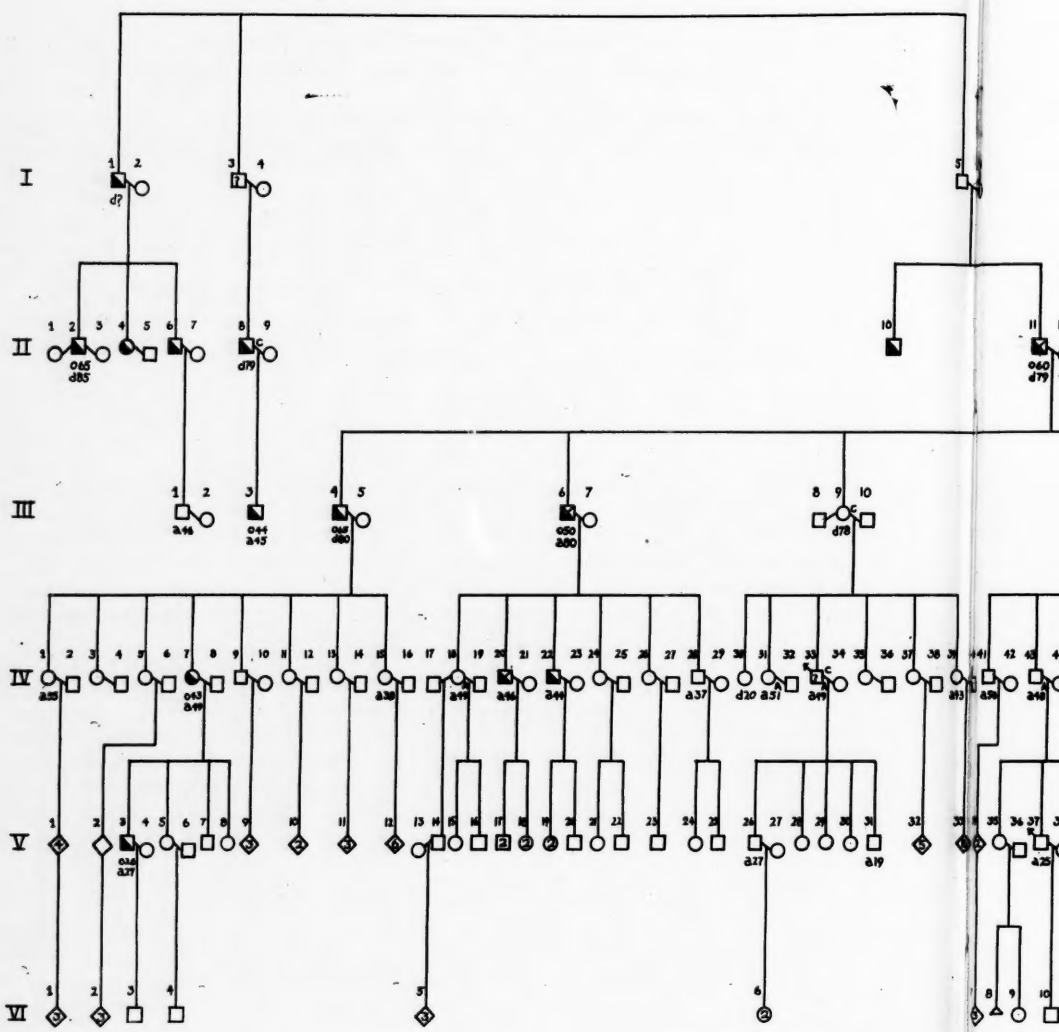
Ataxia is loss of the power of muscle coordination. "The gait presents a coarse ataxic incoordination resulting in a reeling, drunken gait. The reeling is analyzed as being due to lack of synergy between groups of muscles which normally are synergistic. Thus the patient takes a step, but because of asynergy the step is either too long or too short in relation to the intended propulsion of the body, the center of gravity of which either gets ahead of the projected foot or fails to reach it. The misplacement of the foot might be ascribed to dysmetria, a term which is intended to convey the idea that the measure of the step is either too long or too short. If the center of gravity is not projected far enough ahead the patient tends to fall back; if the center of gravity overreaches, the body tends to fall forward."[3]

Ataxia is the result of degeneration of certain nerves. Degeneration of the posterior columns of the spinal cord due to syphilis results in locomotor ataxia. In this family there is no history of syphilis, so it is not the cause of this ataxia. Friedreich's ataxia[4] is a hereditary illness which becomes manifest during childhood. Marie's ataxia,[4] also hereditary, is a cerebellar ataxia. Acute infectious diseases like typhus, pneumonia and rheumatic diseases are among the exciting causes of ataxia. While they are not the direct cause they prepare the system for the degenerative processes which result in ataxia.[3] A sibship in which four of the ten members developed ataxia was reported by Popenoe.[5] In that family the trait behaved as a recessive.

A clinical and genetic study of an ataxic family was made by Waggoner, Lowenberg and Speicher.[8] The 1:1 ratio of affected and unaffected persons was approximated in those generations in which all members had reached the average age of onset. It was suggested that a single dominant hereditary factor is responsible for the transmission of the disease in that family. Fourteen males and 13 females were affected. Since both males and females showed the trait and transmitted it impartially to sons and daughters, the condition was not sex-linked.

Five generations of Marie's hereditary cerebellar ataxia were traced by Oliver and Gray.[4] Unlike many cases of ataxia, mental faculties of affected members were unimpaired. From one ataxic person in generation one, four out of eight children became ataxic. From one affected person in generation two, seven out of nine children, ten out of 56 grandchildren, and one of the 71 great-grandchildren became affected. The age of onset ranged from 21 to 30 years. Oliver and Gray state that others of the grandchildren and great-grandchildren may be expected to become affected as they reach the age of onset. They suggest that the disease might be eliminated from this family if the affected individual did not have children. This would require that individuals having an affected parent should postpone marriage until after they had passed the age of onset of the ataxia.

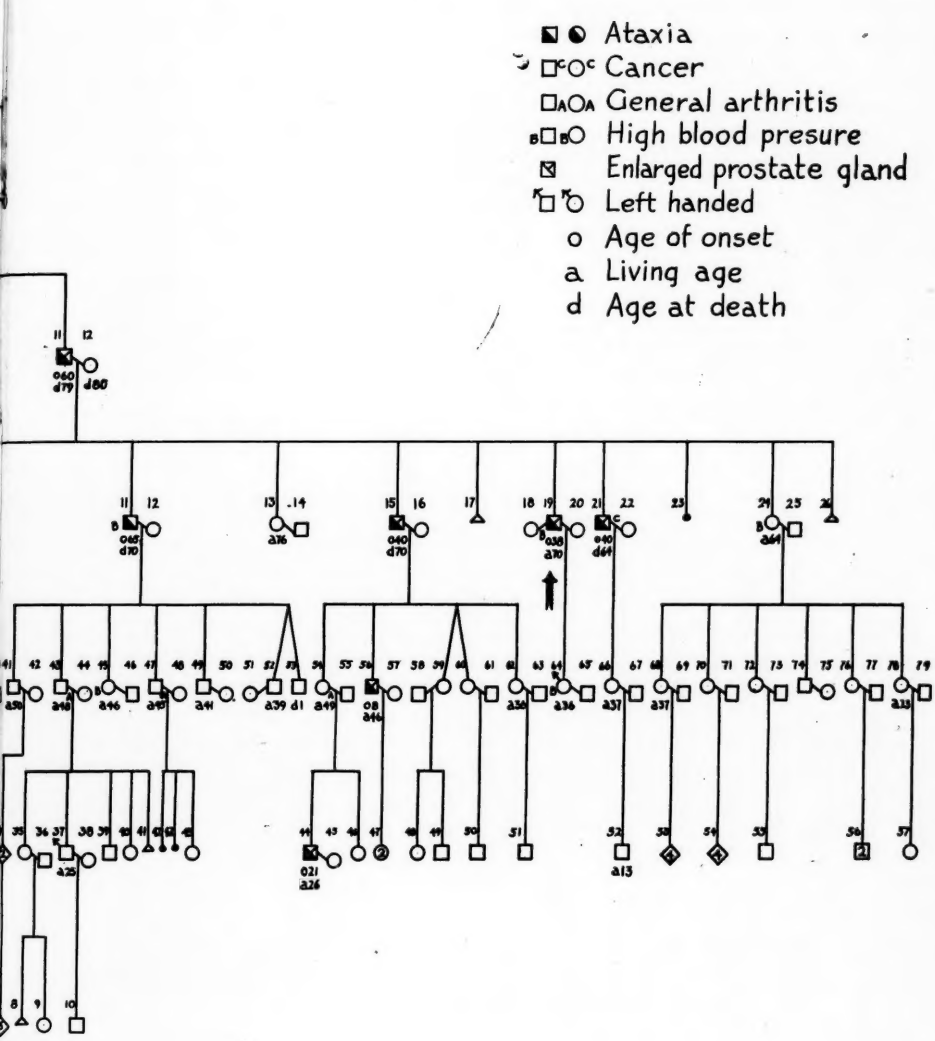
In southern California, Von Hagen[7] reported a case of familial ataxia which he had examined. There was an ataxic



DOMINANT ATAXIA

Figure 7

Six generations of an ataxia different in its symptoms from those previo
nant, with occasional transmission through an unaffected individual (I-5, and
sex-linked character with about 24 per cent of crossing over betwen the *X* an



ATAXIA

ose previously reported. The transmission is in general domi-
(I-5, and IV-54). The ataxia may be inherited as a partially
the *X* and the *Y* chromosomes.

gait, spasticity of the lower extremities and mental deterioration of an unclassified type. A sister became affected at the age of 25. Another sister, who had been affected, was already dead. Three sibs were alive and well.

Milhorat[2] reported hereditary ataxia in two brothers and their sister. Blood and spinal Wassermanns were negative in all three cases. In the males the ataxia was accompanied by muscle atrophy.

A New Ataxic Family

The ataxia reported in the present paper has not been diagnosed as belonging in any of the above classifications. In two persons muscle atrophy is known to be associated with incoordination. Mental status of all affected members is normal. Speech and vision are unimpaired. All the movements of the hands and arms are well coordinated. There appears to be a progressive paralysis of the sensory nerves for pain, since persons affected for many years cease to feel pain.

All the individuals in this pedigree (Figure 7) trace back to three brothers in the first generation. Individual I-1 is the first member of this kinship known to be affected with an ataxic type of walk and stiffness in the knee joints. He had two sons and one daughter who were affected in the same manner. The oldest, II-2, died at the age of 85. His sister II-4 is still living. The younger brother, II-6, had one son, III-1, who is now 46. He is not affected. Information concerning this branch of the family was obtained from him. He is a physician.

Information was not available concerning I-3, but his son II-8 developed an ataxic gait. He died of cancer at the age of 79. His only child, III-3, is a son, now 45 years old. He shows signs of developing the condition. There is stiffness in his knee joints and difficulty in walking.

The third brother in Generation I died young and left two sons II-10 and II-11, both known to be affected. The older son died at an advanced age of unknown causes. The younger son II-11, began to be troubled with stiffness in the knees and severe pain in the legs about the age of 60. This progressed slowly, but paralyzed his legs to such an extent that he was obliged to use a wheel chair during the last few years of his life. He died at the age of 79 from the general depletion of old age and kidney complications. He was the father of 12 children. Three died in infancy, but six boys and three girls reached maturity—to make up the large sibship of Generation III. The six sons all showed the ataxia, but none of the daughters was affected. Since the condition is manifested in three branches of the kinship, the potentiality probably comes from a common ancestor prior to Generation I.

Of II-11's children, the oldest child (III-4) manifested the symptoms slightly. His daughters (IV-5 and IV-7) state that although he was able to walk his gait was ataxic and he dragged one foot. He was 80 years old when he died of a heart ailment.

The second brother (III-6) first noticed difficulty in walking at the age of 50. This was accompanied by severe pain. The knee joints were first involved, becoming swollen, painful and stiff. The condition advanced to the smaller joints of the feet and progressed in the knee and hip joints making it impossible for him to walk. At the age of 80 he is confined to a wheel chair. He has received medical treatment for an enlarged prostate gland.

His sister, III-9, was not affected. She died of cancer at the age of 78. She has five daughters who are not affected. Her son complains of stiffness in his hips, but evidence is not sufficient to include him as ataxic at present.

A brother, III-11, had a history of arrested tuberculosis. There was no other serious illness until he suffered a cerebral hemorrhage at the age of 65. This temporarily paralyzed both upper and lower limbs. Following his recovery from this illness, he had some difficulty in walking. Although the expression of the ataxia in his case was slight as compared to that in 14 other members of the kinship, his son IV-47, who is a physician, considers it is of the same

origin. His walking gait was that of an ataxic. He died of a heart ailment at the age of 70. In his case there was no enlargement of the prostate gland. His five sons and one daughter are not affected. Their ages range from 50 to 39.

A sister, III-13, is now 76. Due to a fractured hip caused by a fall, she is not able to walk well. Her physician attributes this to the injury. Her walk is not typical of an ataxic. She has had no children.

III-15 noted a stiffness of the joints especially the knees, about the age of 40. This increased in severity until he was scarcely able to walk at the time of his death, at 70. The cause of death was an embolism following a prostatomy. His physician said that a greatly enlarged prostate gland was a factor contributing to his inability to walk. He suffered severe pain during the time in which his illness progressed from a slight stiffness to the point where his legs were almost completely paralyzed.

The propositus, III-19, had a history of rheumatic fever as a child. He states that other children in the family suffered from rheumatic and scarlet fever about the same time. Following one of the recurring attacks of rheumatic fever III-19 was unable to walk with ease, and developed a tendency to drag the right foot. At this time he was 38 years of age. The acute manifestations of his rheumatic fever followed the pattern of fever, malaise, and profuse sweating, as described by Lichwitz.[1] Polyarthritis struck, simultaneously involving the legs, especially the knee and hip joints. Stiffness of the muscles, swelling and severe pain in the joints made it almost impossible for the patient to move his legs for a week. The stiffness persisted after the other symptoms disappeared. There was slight cardiac involvement. The patient was cautioned by physicians to use care in the manner and amount of exercise. Subsequent attacks of rheumatic fever followed at the age of 45 and 50. After these attacks the patient found walking increasingly difficult. An enlarged prostate gland received surgical attention at the age of 66. At the age of 70 he is able to walk about the house and dooryard with the use of canes. He has to use care not to fall forward, as his walk is typical of ataxia. There is atrophy of the muscles of the lower limbs. He no longer has pain.

The youngest brother, III-21, had a similar ataxic condition. The illness came on in his early forty's. He suffered severe pain, and in his last years found walking very difficult. He died at 64 following an operation to remove a malignant growth of the prostate gland. This is the only case diagnosed as cancer, although seven other males in the family are known to have had enlargement of the prostate gland.

The youngest sib, III-24, is a woman 64 years old. She works hard and is in good health with the exception of suffering from high blood pressure. Her five daughters and one son are negative in regard to the ataxia. The son is 30 years old, the eldest daughter 37 and the youngest, 23.

To summarize Generation III: there are nine sibs, whose father was affected. Their mother was not affected. Three females are normal in regard to the trait, but all six males are affected. The age at onset of the disease ranged from 38 to 65. The average age of onset for Generation III is 48.

In the fourth generation IV-7 suffered from arthritis and kidney disease. At the age of 43 she developed stiffness in the knees and an ataxic gait. She is the second female known to be affected. Her six sisters and one brother are not affected. The brother is 46 years old. The sisters range from 55 to 38 years in age. IV-7 states that doctors who have examined her are puzzled as to the cause of her disability. There is atrophy of the muscles of the right leg. The kidney condition has cleared up. At 49 she is in good health except for his difficulty in walking. She no longer has pain in her legs.

Individuals IV-20 and IV-22 are also developing difficulty in walking. They are sons of III-6, who is affected. A sister, IV-18, has had arthritis of the spine for eight years. This condition fol-

lowed a severe illness with scarlet fever. Her legs are not affected. Two sisters and one brother are in good health. The brother is 37.

The father of IV-56 was severely affected, and he himself can remember that as a child of eight his knees felt stiff. He could not run and play with ease as other children did. At the age of 36 this condition became much worse. He was treated for an enlarged prostate gland at that time. At the age of 46 he finds walking difficult. One sister has arthritis in her hands but is well otherwise. Her son, V-44, is affected.

The informant, IV-64, daughter of the propositus III-19, is not affected. Her father is affected. She is 36 years of age, and has no children. Individual IV-66 is in good health. Her father was affected. She has one son who is 13 years old.

To summarize Generation IV: three males and one female have ataxia, and 36 individuals are normal with respect to this condition. The age at onset for the female was 43. One boy noted stiffness in his knees at the age of eight, but did not have difficulty in walking until he was 36. The other two males are in their middle forty's and have had the condition for at least two years.

V-3, whose mother, grandfather, and great-grandfather were all affected, is developing stiffness in the knee joints and an ataxic gait. He passed his physical examination for the army and spent several months in service. He was given a medical discharge because he became unable to march due to trouble with his right leg. Physicians were unable to determine the cause of the difficulty. He is now 27 and has been affected about one year.

At the age of 21, V-44 developed stiffness and pain in the legs. Since he knew that his maternal uncle, grandfather and great-grandfather had experienced these symptoms before the onset of the ataxia, he sought medical advice at once. At that time he received medical treatment for an enlarged prostate gland. The prostitis causing this condition was not of venereal origin. The patient's general health and walking ability improved. His parents are not affected.

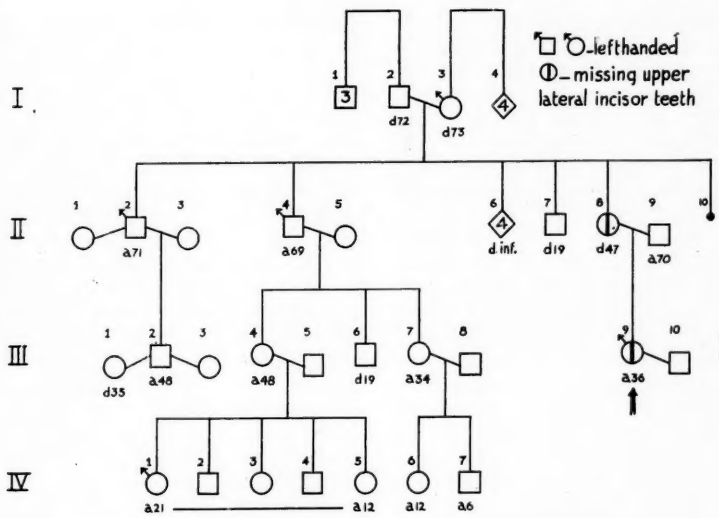
To summarize Generation V: there are two affected individuals and 82 persons who are thus far normal. Others may be expected to become affected as they reach the age of onset. Those who are not affected in this generation range from new born babes to persons 32 years of age. The age at onset for the two affected individuals is 21 and 26.

In Generation VI there are eighteen individuals, nine males and nine females, all under twelve years of age. None is affected.

Discussion

Twenty individuals (18 men and two women) in this kinship are affected with an ataxic type of walk and stiffness in the knees. The pedigree includes 162 persons, 82 males and 80 females, in three branches of the family, and shows six generations. The parents of individuals II-10 and II-11 died at an early age. Had the father I-5 reached the usual age of onset we may expect that he would have developed the condition because his two sons were affected. Since the disease is manifest in one brother, in his three nephews and a niece, the potentiality for developing the trait is probably inherited from a common ancester. The family has lived on this continent since colonial days, but it has not been possible to trace the family history in regard to the ataxia prior to generation I of our pedigree.

Ataxia occurs so rarely in the general population that we can hardly assume that the many individuals who married into the family and had ataxic offspring were heterozygous carriers of a recessive gene. There are no known consanguinous marriages in this kinship. Hence it is not probable that susceptibility to the illness is transmitted recessively through normal parents in any given case. The evidence in generation I is insufficient but in the other 15 cases it is shown that each affected person has one affected parent. In only one case was the condition transmitted from a man (III-15) through his daughter (IV-54) who was



MISSING INCISORS AND LEFT-HANDEDNESS

Figure 8

IV-54 of the ataxia pedigree (Figure 7) is the propositus (III-9) of this pedigree. She and her mother lack upper incisors. Several of her relatives are left-handed.

not affected, to his grandson V-44. If this woman had not produced an affected son it could not be shown that she carried the disease in a latent form.

In this family, with only one exception, each affected person has an affected parent, therefore the condition is due to a dominant gene with penetrance slightly reduced.

There is some evidence in favor of an autosomal dominant with penetrance slightly reduced in the females (IV-54) and some evidence for an incompletely sex-linked gene. "A gene of this nature will behave as an ordinary autosomal factor except for one thing: it will be differentially transmitted to the male and female offspring of a male parent heterozygous for the gene. A male of this sort will tend to transmit such a gene to offspring of the same sex as the parent from whom he received it."[6]

To detect a partially sex-linked gene in this family the critical marriages are: in generation II—6, 8, and 11 (I—1, 3, 5 are the brothers who carry the gene); in generation III—4, 6, 11, 15, 19 and 21. The total offspring are males, affected 10, unaffected 8; females, affected 1, unaffected 18. For the males alone 10:8 is probably too near a 1:1 ratio to indicate linkage and crossing over of the homologous parts of the *X* and *Y* chromosomes. This is evidence in favor of an autosomal dominant. But 10-8-1-18 is certainly not a 1-1-1-1 ratio. However, including both males and females there are 28 (18 + 10) in the non-crossover group and 9 (8 + 1) in the crossover group which would indicate about 24 per cent crossing over. There are two cases in which the man received the gene from his mother but his children are too young to be classified.

In the three families of generation II there are five men and one woman all affected. There are three women in the large sibship of generation III who do not show ataxia. Their six brothers are all affected. Three sibs are unknown.

Their father was obviously not homozygous, and such a distribution is evidence in favor of gene distribution through the *Y* chromosome rather than of reduced penetrance in the females.

The persons in generations IV, V and VI are too young to show symptoms of ataxia, so that they add no critical information.

It has been suggested by physicians attending individuals III-15, IV-56 and V-44 that enlargement of the prostate gland causing pressure on the nerves which supply the legs is a contributing factor. This cannot be the entire cause of the condition since two females show the anomaly, but it may be one of the contributing factors since eight (of the 18) ataxic males have received medical or surgical treatment for an enlarged prostate.

The ataxic gait and stiffness in the knees became so severe in ten cases that it resulted in extreme disability. Two persons became unable to walk. Expressivity of the disease is variable. In two affected members, III-19 and IV-7 the ataxia is accompanied by atrophy of the legs. This atrophy may also be due to variable expressivity.

In this family the ataxia is not due to syphilis for there is no history of syphilis in this kinship. Nor is the gait typical of locomotor ataxia due to tabes dorsalis. Wassermann tests have been negative to the six affected persons known to have been tested.

In seven cases the eldest child is affected. In two additional cases the eldest child is a female, but the second child, the first born son is affected. Ten persons who are affected are not first born children.

The age at onset for Generation III ranges from 38 to 65. In Generation IV the illness became manifest in the early 40's except in the case of IV-56. The two affected persons in Generation V give the age of onset at 21 and 26. Reference to the pedigree chart (Figure 7) would suggest that in successive generations the age of onset of the ataxia appears to be becoming younger. Actually this example of "anticipation" is probably due to three artifacts. First, the oldest member in Generation IV is 55, and in Generation V is 32. Hence a few cases appearing later may yet raise the average of the more recent generations. Second, the absence of clinical records for persons in Generation I, II and III make it necessary for one to rely on the memory of their contemporaries or of their descendants. The age of onset may not be recorded as accurately as in Generations IV and V, where more detailed records are available and all members are still living. Third, members of the younger generations are alert for the symptoms and therefore recognize the disease at an earlier age.

The age at death of the four deceased affected persons in Generation II ranges from 79 to 85; in Generation III the range is from 64 to 80. In Generation IV and V all are still living. It does not appear that the disease shortens the life of affected individuals.

The disease does cause suffering, and in eight cases it resulted in extreme disability. At this time no specific cure or preventive measures are known. Outbreeding has not eliminated the trait, as the family history shows. This ataxia has appeared so late in life that affected individuals have had children and even grandchildren before the condition was recognized. The only method for eliminating the disease in this family would seem to be for individuals in affected families to forego parenthood.

When preventive measures are found, treatment should be started as soon as the hereditary potentiality for developing the disease in the individual can be determined. Even children should be watched for symptoms, since in one case a child was slightly affected. Regular physical examinations of apparently normal persons might reveal premonitory symptoms. More refined nerve or blood chemistry tests might be helpful, but as yet these have not been tried. Thus prophylaxis for the control of the disease might be started before the ataxia is manifest, and known carriers of the disease might forego parenthood, when

the danger of transmitting the disease is recognized.

The variable expressivity of the trait suggests that there are modifying factors, either genetic or environmental. The fact that one woman, who was not ataxic, must have been a carrier because she transmitted the trait to her son, makes it possible to hope that a means of control may be found.

The recessive ataxia reported by Popenoe was manifested at an early age. The autosomal dominant ataxia described by Oliver and Gray appeared before the fourth decade. The ataxia reported by Waggoner et al., also due to an autosomal dominant gene, appeared late in life. Sex does not influence the manifestation of the condition in any of these families. A recessive ataxia, two cases of an autosomal dominant, and now this case which is either autosomal dominant or partially sex-linked in inheritance, are known. It is quite probable that the manner of inheritance may be of aid in diagnosing the kinds of ataxia.

Appendix—Other Traits in the Family

At the time that the ataxia study was made information regarding inheritance of certain other characteristics were also recorded. Some of these characteristics, notably arthritis and blood pressure, may have a direct relationship to the ataxia. The association of the other characters recorded might be significant for linkage studies. Hence these data appear to be worth recording.

Individual IV-18 developed arthritis in the spine following scarlet fever, eight years ago. Her father and two of her brothers are ataxic. Individual IV-33 has stiffness in the hip joints. His sister IV-31 has arthritis in the right shoulder and knee. She states the arthritis followed an infectious condition. These two sibs are children of a normal mother whose father was ataxic. IV-43 developed an arthritic condition following undulent fever ten years ago. His health is much improved and the arthritis has ceased to trouble him His younger brother IV-47, a physician, has had recurrent attacks of arthritis in his left shoulder, hip and ankle, since he was twelve years old. These attacks seem to coincide with sinus attacks. IV-54, whose father, brother and son are ataxic, has arthritis in the hands. In four of the six cases of arthritis the condition has been diagnosed as an infectious arthritis. In some of the ataxic persons the stiffness and pain in the knees has been diagnosed as an arthritic condition. Other physicians do not agree. While the knee joints are involved in the twenty ataxic cases, the condition may not be a true arthritis; therefore the two groups have been discussed separately.

In two members of this family the ataxia is accompanied by muscle atrophy. Individual IV-7 states there is atrophy of the muscles of her right leg. In the case of III-19 there is atrophy of the muscles of both legs. There may be other cases of atrophy which were not reported, but evidence is positive in only these two. This atrophy may be a variable expression of the factor which causes the ataxia.

Data concerning blood pressure was collected for individuals III-11, III-19 and III-24 and their descendants. High blood pressure has been reported in the three persons designated above. IV-45, the daughter of III-II, is receiving medical treatment for high blood pressure. She is 46 years of age. IV-64, the 36 year old daughter of III-19, has blood pressure above normal. The children of III-24 are normal in regard to this trait. They are five females and one male, whose ages range from 23 to 37.

Only four persons in this pedigree are known to have had cancer. Autopsy showed that II-8 died of cancer. III-21 died following surgery to remove a cancerous growth of the prostate. One of his sisters, III-9, had cancer of the face. Her son IV-33 has been treated for cancer of the lip.

It is interesting to note that 138 persons in the third branch of the kinship are right-handed while only three are known to be left-handed. Information was not available in the case of six sibs, children of IV-15, and six infants not yet old enough to determine hand preference. IV-33 is left-handed, as are two of his maternal relatives. Information concerning his paternal relatives is not available. IV-64 is left-handed. Both her parents are right-handed, but two paternal relatives, and two maternal uncles, her maternal grandmother and a maternal cousin are left-handed. She is the propositus in Figure 8. Individual V-37 (Figure 7) is left-handed. His father's cousins IV-33 and IV-64 are left-handed. It is also known that several of his maternal relatives are left-handed. The parents of these three left-handed individuals are right-handed. Therefore the trait must be due to recessive genes, and the parents of the left-handed persons must be heterozygous for the trait.

Individual III-11 had twin boys. His brother III-15 had twin girls. In both cases it is known that multiple births have occurred among the relatives of the mothers, who are not related.

The informant IV-64, who is the propositus III-9, in the pedigree shown in Figure 8, has

a congenital absence of the upper left lateral incisor. Her right lateral incisor is poorly developed. Her mother, II-8 (Figure 8), did not have permanent lateral incisors. Information was not available concerning the maternal grandparents. Since this anomaly was not found among the paternal relatives it would appear that in this case it is inherited as a dominant.

Summary

1. An ataxic gait is manifested in eighteen males and two females, in six generations of a kinship including 162 individuals.

2. Susceptibility has come from a common ancestor prior to Generation I, since the condition shows in three branches of the kinship.

3. Fifteen out of 16 affected individuals each had an affected parent, therefore in this family an autosomal dominant gene appears to be responsible for the susceptibility.

4. Evidence from the critical marriages indicates that it may be a new partially sex-linked gene with a cross-over value of about 24%.

5. One unaffected female transmitted the gene to her son, therefore penetrance is slightly reduced.

6. Expressivity is variable.

7. Eight of the affected males have received treatment for enlarged prostate glands.

8. In this family the ataxia is not due to syphilis.

9. There is no evidence of mental deterioration in any affected members.

10. Eight of the affected persons have lived beyond the age of seventy, therefore the anomaly appears not to reduce the span of life.

11. Other traits found in certain individuals in this kinship are: arthritis, muscle atrophy, cancer, high blood pressure, left-handedness, multiple births, and missing lateral incisors. None of these characters appear to be linked with the ataxia.

12. This ataxia may be eliminated if individuals in affected branches of the kinship refrain from having children. When preventive measures are found, treatment should be started as soon as premonitory symptoms are noted.

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THE SEXUAL HUMORS

THE answers to a number of important problems in biochemistry, embryology, and genetics on the one hand and in clinical medicine on the other lie in the direction of a fuller understanding of the sex steroids. The present volume* contains contributions written from the points of view of all of these various fields. Two symposia are presented in this book; the first entitled "Sex Hormones—Their Actions and Metabolism," comprises papers by Carl R. Moore, A. T. Kenyon, Edward A. Doisy, and F. C. Koch; the second, entitled "Hormonal Factors in the Inversion of Sex," consists of papers by C. H. Danforth, R. R. Humphrey, R. R. Greene, and R. K. Burns, Jr.

To most biologists, who are vaguely aware that the subject of sex hormones is a highly complicated one, it will come as no surprise that this view is shared

*Sex Hormones, edited by F. C. Koch and Philip E. Smith. Biological Symposia, vol. IX; X + 146 pages; $2.50; The Jaques Cattell Press, 1942.

by workers in the field. Among outstanding problems which the reader will find discussed here are the source of hormones produced by castrates (there are good reasons for suspecting the adrenals), the origin of androgens in female urine and of estrogens in male urine, and the physiological basis of the frequently observed heterodox, or nonspecific, action of the hormones. On the chemical side are problems of basic technique, such as the development of specific methods for the determination of various classes of steroids, and the improvement of existing methods for liberating the free hormones from the conjugated forms which occur in the urine.

Of particular interest to geneticists is the section dealing with the inversion of sex. It is possible to produce more or less complete somatic inversion of vertebrates, starting with either sex, by the administration of hormones, either as crystalline compounds or, in some instances, through grafting procedures. It is generally agreed that the time element is a deciding factor in determining the extent to which inversion may occur. The most complete inversions are obtained by the treatment of embryonic stages, before the irreversible loss of structural rudiments of the opposite sex has taken place. Treatment of adults of the higher vertebrates leads to little or no morphological change except, as Danforth points out, in the case of regenerating parts (e.g., feathers) where the structure passes through an effectively embryonic stage at each regeneration. In Amphibia the persistence of gonadal rudiments into larval life makes possible the total inversion of sex. According to Humphrey, the inversion process in Amphibia occurs in two stages: first, the suppression of the normal gonad, and second, the growth of the rudiment into a functional gonad of the opposite sex.

The spectacular inversions which can be obtained with suitable choice of organism and hormone make it all the more necessary to explain the not uncommon paradoxical effects—i.e., feminizing effects of androgens and masculinizing effects of estrogens. A few typical examples gleaned from the present work are the feminizing effects of testosterone on plumage in Phasianidae and on Mullerian duct derivatives of the opossum, and the masculinizing influence of estrogens on the Wolffian duct of female rats. Although the problem is not solved, a number of interesting hypotheses are proposed: the embryonic gonadal hormones which actually control sexual differentiation differ from the known adult sex hormones (Greene); the paradoxical effects result from the unphysiologically high dosages of hormones usually used in experiments (Burns); sex hormones have nothing to do with embryonic sex differentiation (Moore).

Bibliographies are provided at the end of such paper, but there is no index.

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A GENETIC ANALYSIS OF THE AMERICAN QUARTER HORSE

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THE American Quarter Horse or the American Quarter Running Horse is classed by some to be the first American horse breed. Michaelis[5] credits it with being an established breed in 1665. However as late as 1936 Willams and Jackson[7] refer to it as a "type" which was developed upon the western range to serve as a cow horse. Considerable uncertainty has existed as to the true relationship of the Quarter Horse and the Thoroughbred. Very widely separated opinions are held in this regard. Quarter Horse breeders freely admit the practice of occasionally crossing to the Thoroughbred. Denhardt[2] asserts that "the American Thoroughbred was founded with the help of the Quarter Horse."

In 1940 the American Quarter Horse Association was organized. This association has since begun the publication of a *Quarter Horse Stud Book*, of which the first instalment (Volume One, Number One) appeared in 1941.

It was thought that the information made available in this stud book might establish certain facts in regard to the structure and genetic history of the breed. The questions which it was hoped to answer were: (1) How much inbreeding had been practiced in producing the animals being selected to found a pedigreed breed? (2) How much relationship existed between those animals which have been bred over a long period without having to adhere to limits set by any stud book? (3) Are there any individual animals which have been of outstanding importance in the breeding of these foundation animals? (4) How great has been the influence of the Thoroughbred upon the evolution of the Quarter Horse? (5) What has been the average length of time between generations in this breed?

Scope and Methods

The methods used in this study were those developed by Wright[8] and first applied to breed analysis by McPhee and Wright[4] in their work on the British Shorthorn. Since that time Calder[1] has applied the method in his study of the Clydesdale breed of horses in Scotland, as did Steele[6] in his study of Thoroughbred, Standardbred, and American Saddle horses.

Originally we intended to include in this study only the 535 animals listed in Vol. I, No. 1 of the *Quarter Horse Stud Book and Registry*. However, before any great number of pedigrees had been tabulated, Vol. I, No. 2 was published. The plan for this study was altered to include animals from both numbers of the *Stud Book*. The plan was revised to include three samples of the breed based on date of birth of animals included. One sample comprised the 126 animals foaled prior to 1930. Animals for this group were recorded in both numbers of the *Stud Book*. The second sample selected included the 127 animals foaled in 1935. The third sample was made up of the 122 animals foaled in 1940 and 1941, the foals for two years being included in order that the three samples be of approximately the same size.

Since considerable work had already been done on animals from Vol. 1, No. 1, a sample from this material was included in the study. This consisted of the ninety stallions, regardless of birth date, which appeared in the first number of the *Stud Book*. Preliminary study of the first ninety mares in numerical order of appearance in the first volume of the *Stud Book* revealed that this group was composed largely of animals bred by the King Ranch. There

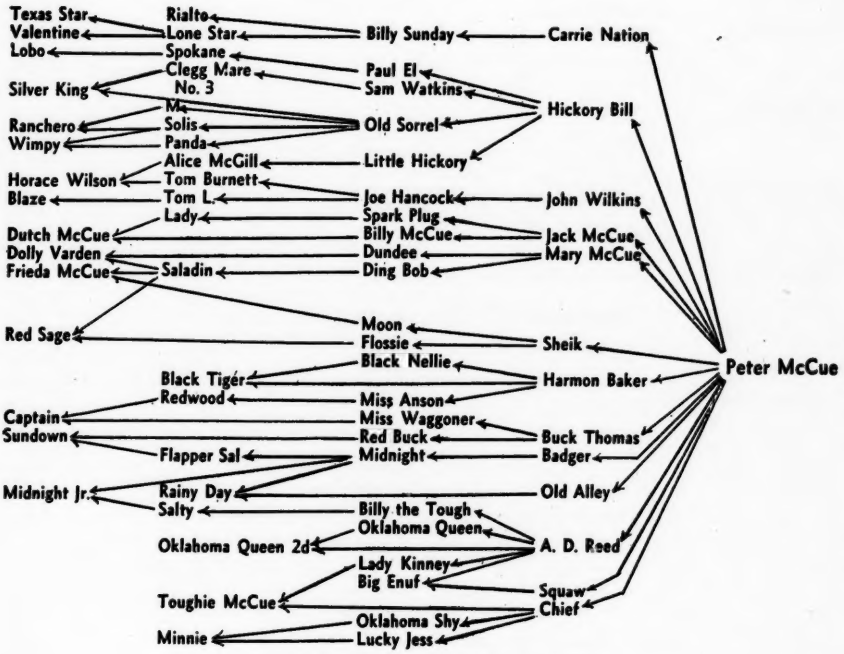


Figure 9

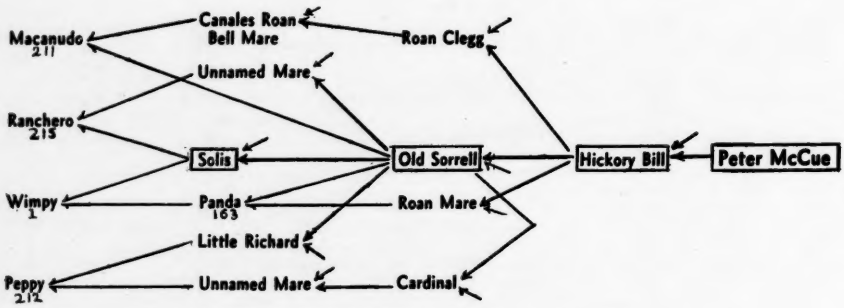
Lines of descent of some of the animals tracing back to Peter McCue. Peter McCue is the most important sire in the Quarter-Horse breed. His son Hickory Bill is also among the four leading sires.

seemed to be a decided difference in the breeding of the King Ranch individuals, and to check this point another sample was included, composed of the 118 animals which had been bred by the King Ranch. As a result there is considerable duplication in certain of the samples. Some of the animals from the King Ranch sample and the ninety-stallion sample were included in the breed samples. However, the number of these animals included in any breed sample was not large. In fact, the rather uniform number of duplications in the various samples was taken to indicate that they were comparable, and representative of the breed.

Four-generation pedigrees were tabulated for each of the animals in the study. These pedigrees were complete in very few cases because in many instances the breeding behind some animal in a pedigree would be unknown. Also a Thoroughbred often appeared close up in the pedigree. In such cases no attempt was made to carry the ancestry back of the Thoroughbred animal. The inbreeding and relationship coefficients were determined entirely from these four-generation pedigrees. Even when it was obvious that extension of a pedigree would yield a higher coefficient of inbreeding or relationship, the pedigrees were not extended beyond the four generations.

Inbreeding

Table I shows the coefficients of inbreeding for the five samples. The three samples designated: "Before 1930,"



ANCESTRY OF KING RANCH STALLIONS

Figure 10

Lines of descent of four King Ranch stallions showing relat.onship to Old Sorrel, Hickory Bill, and Peter McCue.

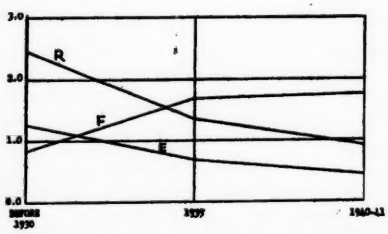
"1935," and "1940-41" are considered representative of the breed. It is noteworthy that the "Stallions" sample, in spite of the high proportion of King-Ranch-bred animals, differs little from the breed average. The lower figure for this sample is most probably explained by the relatively greater number of unknown lines in these pedigrees. It is believed that the figure of approximately 1.7 per cent inbreeding as it occurs in the other three samples is nearer the true inbreeding coefficient for the breed.

With the exception of the "Before 1930" category, the actual inbreeding in the four representative samples is greater than the calculated or expected inbreeding. However this difference hardly appears great enough to indicate any appreciable amount of planned inbreeding. It is surprising that the difference is no greater since there have been no breed limits to build relationship in the Quarter Horse.

The inbreeding coefficients of 1.7, 0.86, 1.64, and 1.75 per cent found in our four samples are considerably lower than the average coefficient of 6.5 per cent for the Clydesdale breed as reported by Calder.[1] However, the higher coefficient for the Clydesdale was attained over a sixty-year period. This is a slightly longer period than would ordinarily be covered by the four generations from which the Quarter Horse coefficients have been obtained. Furthermore coefficients found in one sampling are very similar to those given by Steele[6] for selected groups of Thoroughbreds, Standardbreds, and American Saddle horses. He reported a coefficient of 1.4 per cent for the Thoroughbreds, 1.6 per cent for the Standardbreds, and 2.8 per cent for the American Saddle horses. All were based upon complete five-generation pedigrees.

Inter se Relationship

The average *inter se* relationship coefficients for the several samples are shown in Table II. It has already been pointed out that these coefficients are too small to account for the inbreeding of the corresponding samples. The small amount of *inter se* relationship may be partially attributed to the lack of information as to the ancestry in many pedigrees. However, the *inter se* relationship progressively decreases from the older to the more recent samples, at the same time the pedigrees are becoming more complete. Thus the relatively sketchy pedigrees of "Before 1930" show a relationship of 2.46 per cent. In the 1940-41 sample, with the most complete pedigrees, the relationship is only 0.98 per cent (see Figure 11). This trend in relationship and inbreeding may mean that there has been some separa-



INBREEDING AND RELATIONSHIP COEFFICIENTS

Figure 12

Average actual inbreeding (F), *inter se* relationship (R), and inbreeding expected from *inter se* relationship (E) are shown for the three periods sampled. The actual inbreeding is higher than expected, but not enough to indicate a deliberate plan to inbreed to certain favored ancestors.

tion into family lines in the animals giving rise to the *Stud Book* of the Quarter Horse breed.

Outstanding Individuals

Certain animals usually assume particular importance in the ancestry of any breed of livestock. The Quarter Horse is no exception, although at present only a small number of animals have achieved decided prominence. In Table III are listed the most frequently occurring sires in these four-generation pedigrees. The number of times each of these animals appeared in each sample shows that during the period covered only six are of very great importance. Of those six sires, four appear with about the same frequency. In all samples none of these foundation animals referred to in this discussion is included in the *Stud Book*. They are therefore designated by name without a registration number. The four outstanding sires are Peter McCue, Hickory Bill, Traveller, and Little Joe. Hickory Bill is a son of Peter McCue and Little Joe a son of Traveller. All but one of the remaining animals in the list trace to Peter McCue or to Traveller. King is a full brother to Little Joe. Harmon Baker and Badger are sons of Peter McCue and Old Sorrell and Billy Sunday are grandsons. Sykes Rondo is the Grandsire of King and Little Joe through their dam. Reference to Figure 9 shows more clearly the relationship of these animals tracing to Peter McCue.

Two facts to be derived from Table III indicate that Peter McCue has been the most important sire in the breeding of the Quarter Horse. The first fact is the decidedly greater number of times he appears in the pedigrees. The second is that he far exceeds either of his sons in number of appearances. At the same time there is little difference to be found in the number of appearances of Traveller and his son Little Joe. As Traveller had other sons it appears that he occurs primarily as a result of a choice by breeders of animals tracing to Little Joe.

Table IV presents a different arrangement of the data from Table III. In this table are shown the direct relationships to each sample of the breed of the six sires occurring most frequently. This brings to a more comparable basis the differences in frequency of occurrence as an ancestor resulting from difference in time of birth. That is, an animal born at a later date would normally have fewer opportunities to appear as an ancestor, but because of later birth would be more closely related to those animals tracing to him. The information in this table again demonstrates the importance of Peter McCue. The relationship of Peter McCue to the various samples shows less variation than that of the other sires listed. In addition, his relationship of 5.68 per cent to the 1940-41 sample is only slightly less than his 6.35 per cent relationship to the "Before 1930" sample. In each of the three samples based upon year of birth, Peter McCue's relationship is greater than that of his son Hickory Bill. Little Joe is more closely related to each of these three samples than is his sire, Traveller. The average relationships of Hickory Bill and of Little Joe to these three samples are 3.77 per cent and 3.74 per cent respectively.

King Ranch Breeding

As previously stated, a casual survey indicated that the breeding methods employed by the King Ranch differed markedly from those generally employed by Quarter Horse breeders. This conclusion is confirmed by the inbreeding coefficients for King Ranch horses presented in Table I. The average coefficient of 4.89 per cent is more than twice the coefficients for the representative samples of the breed. This is a relatively low coefficient when compared to that of 40.9 per cent found by Wright[9] for the Shorthorns bred by Thomas Bates. It does not mean that radically close inbreeding has been practiced.

It will be noted from Table I that the expected inbreeding for the King Ranch sample is more than twice the actual inbreeding. This is the opposite of the condition found for most of the other samples. We interpret this to mean that among the animals bred at the King Ranch a deliberate effort had been made to reduce the inbreeding below that to be expected if random mating had occurred within the group. Closer study of the pedigrees of the animals revealed another possible explanation. A considerable number of animals in the sample were the result of cross-breeding to Thoroughbreds. This meant that these animals, though not inbred, were still closely related through the Quarter Horse side of the pedigree.

Table III shows that only three of the frequently occurring ancestors in the other samples are prominent in the King Ranch sample. In this sample Hickory Bill was the most frequently appearing sire, followed by his son Old Sorrell. This indicates that in the breeding of these horses Peter McCue appears as an ancestor due to the frequent use of his son and grandson. A composite pedigree of recent King Ranch sires (Figure 10) shows how relationship to Old Sorrel and Hickory Bill has been maintained. This is typical of the breeding of other King Ranch animals, and indicates the manner in which the increased coefficient of inbreeding of these animals has come about.

The fact that the frequent appearance of Peter McCue as an ancestor was incidental to line breeding to his son and grandson is again brought out in Table IV. The direct relationship coefficient of Peter McCue is roughly one-fourth the 40.29 per cent relationship of Old Sorrel. The 21.45 per cent coefficient for Hickory Bill is approximately one-half that for Old Sorrel. The fact that Hickory Bill was slightly more than one-half as much related as his son indicates that he appeared in the ancestry through other lines than as the sire of Old Sorrel. Perhaps the most important fact shown by Tables III and IV is that the King Ranch strain was bred almost entirely from Peter McCue and his offspring and practically not at all from Traveller, the other major line used by the average breeder.

Thoroughbred Influence

Reference to the use of Thoroughbreds by Quarter Horse breeders has already been made. The Thoroughbred sires which had been used most extensively are listed in Table V. The number of times each appeared in the pedigrees of animals in each sample is also shown. It is of interest that some of these Thoroughbreds occurred as often as some of the foundation Quarter Horse sires included in Table III. For example Horace H and Bonnie Joe each appeared at least once in each of the five samples. Horace H was the sire of the Quarter Horse Billy Sunday listed in Table III. Derring-Doe and Chicle also appeared in four of the five samples.

There are no mares listed in Table V even though several were observed a number of times in the pedigrees. One of these frequently occurring Thoroughbred mares was Zepplin. Perhaps the mare occurring most frequently was Nora M. She was not included in the list of frequently occurring animals because she appeared only as the dam of Peter McCue. In the light of the important place Peter McCue has a foun-

dation sire the influence of the Thoroughbred would be considerable through this single mare. In the same manner, the influence of the Thoroughbred might be rather great as the result of Old Sorrel's dam.

The Thoroughbred stallions mentioned above are by no means the only ones appearing in Quarter Horse pedigrees. All of those appearing will not be listed, but the names of a few additional ones might be of interest: Spearmint (the sire of Chicle), Primero, Dundee, Leadville, Lovely Manners, Ben Brush, and Man o'War.

Not all of the Thoroughbred animals appearing in the Quarter Horse pedigrees were identified. In many instances there was the simple notation in the pedigree that the sire or dam of an animal was a "Thoroughbred." Hence a survey of the known Thoroughbred ancestors does not give a complete picture of the influence of the Thoroughbred. The number of animals in each sample tracing to an identified or an unidentified Thoroughbred is shown in Table VI. This shows that about 55 per cent of the Quarter Horses entered in the *Stud Book* sample traced to at least one Thoroughbred ancestor. It is also shown that 97.4 per cent of the horses bred by King Ranch had Thoroughbred ancestors within the first four generations. This condition shows that King Ranch has made considerably greater use of the Thoroughbred in its breeding program than has been true with most Quarter Horse breeders. This extensive use of this breed by King Ranch alone gives an important place to the Thoroughbred. Approximately nine per cent of all the Quarter Horses accepted for registration at the time of this study has been bred by King Ranch.

Interval Between Generations

The interval from one generation to the next is of importance in the breeding of any type of livestock. The findings in regard to this point for the Quarter Horse are shown in Table VII. The amount of information available regarding generation intervals was somewhat limited. This was the result of the fact that in the *Stud Book* birth dates of the animals were available only in cases where a registered animal had at least one parent registered. The data for Table VII were secured from the animals in Vol. I, No. 2 meeting this criterion. The average interval between parent and offspring was found to be 8.99 years. The average interval from sire to offspring was 9.56 years and from dam to offspring 7.91 years. This average generation interval of 8.99 years was somewhat shorter than that reported by Steele.[6] He found that the interval between generations was slightly over 12 years for the Thoroughbred and slightly over 11 years for the Standardbred and American Saddle Horse. The shorter interval between generations found in the Quarter Horse may be due to the fact that outstanding sires have not played so important a part in the breed. Thus no particular effort has been made to keep stallions in service over extremely long periods.

The interval between animals born in 1940 and their parent's birth is also shown in Table VII. This differs little from that found for animals born at earlier dates. It appears that there has been no change in the average interval between generations in recent years.

It is interesting to observe one difference in the interval between generations in the Quarter Horse and the interval between generations in Holstein-Friesian cattle as reported by Lush *et al.*[3] Lush and associates found the average interval between the dam and calf was 5.3 years while between the sire and calf was only 4.2 years. The reverse situation found in the Quarter Horse has already been pointed out. In this breed the interval from sire to foal exceeded the interval from dam to foal. These intervals were 9.46 years from sire to foal and 7.91 years from dam to foal. This seems to point to a relatively more extended use of a few chosen sires in the Quarter Horse than has been the practice in Holstein-Friesian cattle.

Summary

The findings of this group of the breeding of American Quarter Horses are as follows:

1. Analysis of representative samples of the animals accepted for registration showed the coefficient of inbreeding to be approximately 1.7 per cent.

2. The coefficients of relationship within representative samples ranged from .98 to 3.02 per cent.

3. The most frequently occurring ancestor in the samples as a whole was Peter McCue who showed a direct relationship of 5.68 per cent to the animals born in 1940 and 1941.

4. Animals bred by the King Ranch differed substantially from the breed as a whole. The coefficient of inbreeding for these animals was 4.89 per cent and the *inter se* relationship was 20.11 per cent. The King Ranch horses showed a direct relationship of 6.69 per cent to Peter McCue and 40.29 per cent to his grandson Old Sorrel.

5. The influence of the Thoroughbred is demonstrated by the fact that more than 50 per cent of the animals in the study traced to at least one Thoroughbred ancestor. In the King Ranch sample 97 per cent traced to at least one Thoroughbred ancestor.

6. The average interval in years between generations was found to be 8.99 years. The interval between dam and foal was 7.91 years and between sire and foal 9.45 years.

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Table I. - Average Coefficients of Inbreeding

Sample	Number of animals	Actual inbreeding %	Expected inbreeding %
Stallions	90	1.70	1.52
Before 1930	126	.86	1.25
1935	127	1.04	.67
1940-41	122	1.75	.49
King Ranch	116	4.89	11.10

*Calculated from relationship in Table II.

Table II. - Inter se Relationship Coefficients

Sample	Number of pairs	Inter se Relationship %
Stallions	45	3.01
Before 1930	63	2.46
1935	63	1.34
1940-41	61	.98
King Ranch	58	20.11

Table III. - Number of Appearances of Frequently Occurring Quarter Horse Ancestors

Ancestor	Sample				
	Stallions	Before 1930	1935	1940-41	King Ranch
Peter McCue	70	85	90	84	147
Hickory Bill	34	30	33	30	176
Herman Baker	12	4	11	19	-
Badger	4	4	10	6	-
Old Sorrel	14	20	20	9	166
Billy Sunday	4	3	15	8	-
Traveller	24	28	42	28	6
Little Joe	17	13	32	25	3
King	4	5	10	10	-
Sykes Ronde	18	23	22	5	2

Table IV. - Direct Relationship to Selected Ancestors

Ancestor	Sample				
	Stallions	Before 1930	1935	1940-41	King Ranch
Peter McCue	8.19	6.35	4.68	5.68	9.89
Hickory Bill	5.56	5.15	3.98	3.20	31.45
Old Sorrel	5.69	7.34	4.68	1.07	40.29
Traveller	2.91	3.42	3.10	1.64	.42
Little Joe	3.96	3.77	5.14	2.30	.53
Sykes Ronde	1.25	1.56	1.08	.25	.10

Table V. - Frequently Occurring Thoroughbred Ancestors

Ancestor	Sample				
	Stallions	Before 1930	1935	1940-41	King Ranch
Bonnie Joe	5	4	1	7	2
Chisle	1	-	2	1	10
Darring-Doe	-	2	4	6	1
Horace H.	2	3	10	2	1
Right Royal	2	9	-	-	30
Whisk Broom II	1	-	7	-	1

Table VI. - Quarter Horses with Thoroughbred Ancestors within Four Generations

Sample	Number of animals	Number with Thoroughbred ancestors	Percent with Thoroughbred ancestors
Stallions	90	49	54.4
Before 1930	126	70	55.5
1935	127	70	55.1
1940-41	122	70	57.3
King Ranch	118	115	97.4

Table VII. - Interval in Years per Generation

Date of birth of offspring	Type of Interval	Number of Intervals	Interval in Years
1940	Sire to foal	68	9.70
	Dam to foal	36	7.98
	Average	104	9.10
1940 and before 1940	Sire to foal	286	9.46
	Dam to foal	423	7.91
	Average	409	8.99

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